Muscle Paralysis in Herpes Zoster

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■ *Herpes zoster may, in some instances, cause motor paralysis as well as the usual sensory and cutaneous manifestations. It is suggested that the presence of electromyographic denervation potentials be used as the criterion of muscle paresis in order to avoid mistaking atrophy of disuse for true lower motor neuron disease. Use of the proper physical therapy procedures hastens the recovery of function and may serve to retard denervation atrophy and fibrosis in patients with muscle paralysis.*

HERPES ZOSTER, or shingles, is an acute infectious disease affecting man only. The causative agent is herpesvirus zosteri, a member of the herpesvirus group which all have an affinity for ectodermal tissues.[16] The morphologic and physical properties of the zoster virus are identical with those of the causative agent of chickenpox.[16] Epidemiological observations, serological studies and cultural characteristics also confirm the opinion of most virologists that the two diseases are caused by the same organism.[16,20]

The disease characteristically begins with itching followed by pain radiating into the distribution of a spinal or cranial sensory nerve. After a few days a vesicular rash appears in the cutaneous distribution of the sensory portion of the involved spinal root. At this point, the diagnosis becomes apparent from the peculiar combination of pain and eruption localized to a single dermatome or to adjacent dermatomes. Some variations may occur —for example, the eruption may precede the pain or pain may not occur or there may be signs of meningeal irritation. In most cases the pain subsides and the eruption heals, leaving a slightly pigmented scar. The total course of the illness is about five to eight weeks. The most common complications are secondary infection in the skin and persistent post-herpetic pain.[4,6,16] Correlating with the clinical manifestations, it has been shown that the virus can be recovered from a skin lesion and can cause an inflammatory reaction in the sensory ganglion, dorsal root and posterior horn of the spinal cord.[16]

There are rare cases of herpes zoster that do not fit the typical features and in which widespread involvement shows that the disease can be a generalized infection of the central and peripheral nervous system.[13] As examples, flaccid muscle paralysis can occur* due to spread of the infection from the posterior horn of the spinal cord to the

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anterior horn and the motor nerve root.[1,8] Lower extremity spastic paralysis can be caused by interruption of pyramidal tracts in the spinal cord.[11,13] Cranial nerve involvement may cause paralysis of extra-ocular muscles[4] or facial paralysis as in the Ramsay Hunt syndrome.[5,10,18] Diaphragmatic paralysis can occur when cervical nerve roots are involved.[9,14] Upper motor neuron signs together with disturbances of consciousness can signify encephalitic involvement.[17]

It is these unusual types of herpes zoster, in which the motor system is involved, that come to the attention of the physiatrist, orthopedist, neurologist or other physician specializing in musculoskeletal disorders. We have recently had three patients with such cases under our care, and in addition we reviewed the records of ten other cases at the Los Angeles Veterans Administration Hospital.

Reports of Cases

CASE 1. The patient, an 89-year-old Caucasian man, a Spanish-American War veteran, living in Southern California, was in good health until mid-August 1964, when pain developed in the right thigh and was followed by a vesicular rash. A diagnosis of herpes zoster was made by his physician. The pain continued and the patient complained of poor appetite dating from the onset of the disease. About two weeks after the pain and rash appeared, weakness of the right lower extremity began, and gradually progressed to the point where the knee would buckle on ambulation. Five weeks after the disease began, the patient was admitted to the Veterans Administration Hospital because he was unable to walk. He also complained of persistent and severe pain localized to the medial side of the right knee.

About a year before admission, the patient had a transient episode of right facial weakness, lasting one day, which was felt to be unrelated to his present disease. There had been no recent injuries. The patient was under treatment for conjunctivitis for the preceding six months and had been receiving digoxin. He did not remember having had varicella and did not know of any recent contacts with it.

On examination, light brown scars of herpetic eruption were present in two strips on the right buttock and on the right anteromedial thigh. (See Figure 1.) Musculature of the right thigh, particularly the vastus medialis, appeared atrophied but

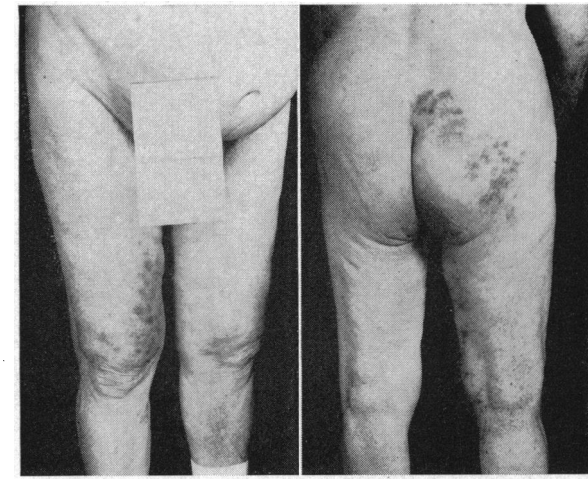

Figure 1 (Case 1).—Photographs showing the distribution of healing vesicular rash about six weeks after onset of disease.

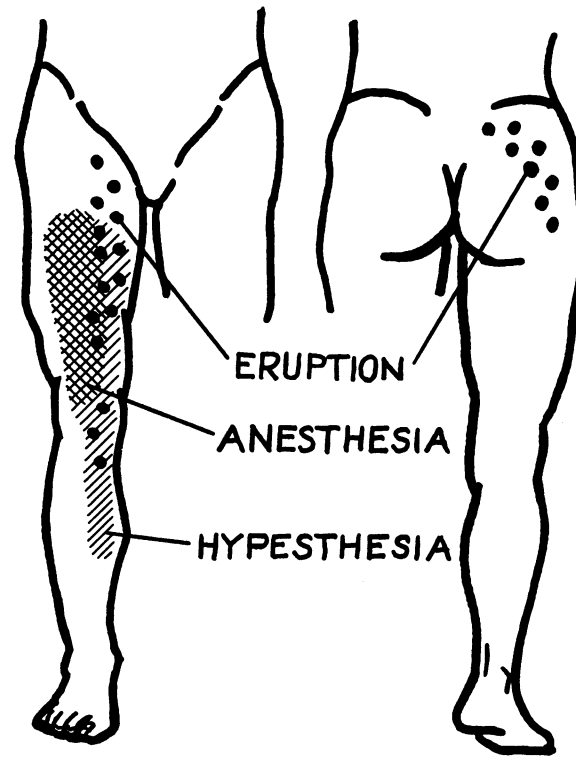

Figure 2 (Case 1).—Diagram showing the distribution of sensory loss to pinprick in relation to the location of the eruption.

the legs were of equal circumference at the thighs and calves. Pronounced weakness in the right lower extremity was found on manual testing of the muscles, the proximal muscles being weaker than the distal. Specific muscle group ratings were as follows: hip flexors, poor; hip adductors, fair; hip abductors, fair; quadriceps femoris, poor; foot dorsiflexors, good; foot plantarflexors, good. Deep

tendon reflexes were depressed equally in knees and ankles bilaterally. There was an area insensible to pinprick on the anterior aspect of the right thigh from mid-femur to the patella, and medially there was a larger area of decreased sensation (Figure 2). No impairment of sensation could be found in the buttock, the back of the leg or the foot. Electromyographic examination showed denervation potentials consisting of fibrillation and positive sharp waves in the quadriceps, anterior tibial, peroneus longus and gastrocnemius muscles. X-ray examination of the lumbosacral spine showed advanced degenerative changes with large vertebral osteophytes.

A physical therapy program was prescribed, consisting of range of motion exercises of the right lower extremity joints to prevent contractures, progressive resistance exercises to strengthen the weakened musculature and galvanic electrical stimulation to help preserve denervated muscle while awaiting regeneration of the involved nerves. After ambulation training the patient was able to walk with the help of a pick-up walker in spite of only a small increase in strength in the paretic limb. At no time requiring more than salicylates, the pain gradually subsided over approximately three months. About four months after the onset of the disease the patient was discharged to the care of relatives out of town. The final outcome of the paralysis was not ascertained.

CASE 2. A 44-year-old woman bookkeeper had at first an itching sensation over the right upper extremity and then severe pain and a vesicular rash over the distal arm, dorsal aspect of the forearm and the ulnar aspect of the hand in the area of dermatomes supplied from the seventh and eighth cervical nerves. Weakness of the triceps, ulnar wrist extensors, the extensor pollicis longus, opponens pollicis, the interosseus muscles and the ulnar-innervated lumbricals became evident as the rash began to subside. The patient was unable to write or to use her hand because of pain and weakness.

Electromyographic examination six weeks after the onset of the illness revealed fibrillation of denervation activity in the muscles of the right upper extremity supplied by the anterior primary ramus of the eighth cervical nerve root. The denervation activity was most pronounced, quantitatively, in the pure C8-innervated area (flexor carpi ulnaris, dorsal interossei and adductor pollicis muscles).

Treatment consisted of whirlpool ultrasound and neuromuscular reeducation techniques. Electrical stimulation was added a week later to retard denervation atrophy. Pain regressed slowly and limited function returned. It was believed that the patient would require a fairly long program of physical therapy for optimum functional recovery.

CASE 3. A 68-year-old housewife had first an itching sensation over the right ear and face and then a vesicular rash over the outer surface of the ear. Pain in the ear, the face, and the right side of the neck developed three days later along with obvious weakness of the right facial muscles.

On examination six weeks later, incomplete paralysis of the right facial musculature was noted. The eyelid could not be completely closed, the patient was unable to whistle and there was absence of spreading of the orbicularis oris muscle on the right side. Sensitivity of the skin over the external ear was also present. Electromyographic examination showed moderate, partial denervation of the right frontalis, nasalis and orbicularis oris muscles.

A diagnosis of herpes zoster involving the right facial nerve (Ramsay-Hunt syndrome) was made on the basis of the history, clinical findings and electromyographic examination.

Physical treatment, begun immediately, consisted of electrical stimulation, massage and resistive exercises. Progressive improvement in muscle strength has been evident.

Study of Case Records

Our interest aroused by these three cases and further stimulated by an excellent review of the records of cases at Massachusetts General Hospital by Grant and Rowe,[8] we undertook a review of the case histories of all patients at the Veterans Administration Hospital, Los Angeles, who had a diagnosis of herpes zoster during the period 1953-1964. In many of these cases the zoster was not the primary diagnosis, and often, with or without motor paralysis, it was an incidental finding and not carefully studied. None of these patients were seen by the authors and only chart notes were available for evaluating the cases.

There were 208 patients with a diagnosis of herpes zoster in this group. In ten cases there were sufficient findings recorded to make a reasonably certain diagnosis of motor paralysis due to the zoster. Unfortunately, electromyography was not

TABLE 1.—*Anatomic Location of Herpes Zoster in Cases Reviewed from Records of the Veterans Administration Hospital, Los Angeles, 1953-1964*

Location	*All Cases*	*Paralytic Cases*
Brain (encephalitis)	1	0
Scalp & Neck	7	0
Cranial Nerves	51	8
Upper Extremity	21	0
Trunk	91	1
Lower Extremity	30	1
Generalized	2	0
Unknown	5	0
	208	10

done in any of these cases for positive evidence of lower motor neuron disease. The age range of the patients was similar to that noted in previous reports.[8] The anatomic sites of the zoster infection are shown in Table 1. The table does not include the three cases with which the present report began. Brief summaries of the ten paralytic cases found in the histories studied are given below.

CASE 4. The patient was a 65-year-old Caucasian man with diabetes. A herpes zoster eruption developed over the left side of the face on the forehead, eyelid, cheek and nose. It had been present for four days when the patient was admitted to the hospital May 20, 1953. Conjunctivitis of the left eye was also present. Left upper lid ptosis, enophthalmus, and extra-ocular muscle paresis gradually developed. The patient was discharged five weeks later in an improved condition to be followed as an outpatient for unrelated conditions of arteriosclerotic chronic brain syndrome and peptic ulcer. Corneal keratitis developed in the left eye and four months later he was readmitted for tarsorrhaphy. The ocular movements at that time were believed to have returned to normal.

CASE 5. A 56-year-old Caucasian woman was examined by the ophthalmological consultant on January 5, 1955 and found to have left fourth cranial nerve paresis probably due to herpes zoster involving the skin about the eye. An eye patch for diplopia was recommended. No further information is available.

CASE 6. A 74-year-old Caucasian woman was admitted to the hospital October 15, 1958 with complaint of nocturnal radicular pain and blistering in the dermatomes on the right side supplied from the twelfth thoracic and first lumbar roots. This condition had been present for ten days. A diagnosis of herpes zoster was made. The skin lesions responded to local treatment and the patient was discharged in two days. On readmission two months later the examining physician noted that, with the patient standing, there was a slight bulging of the right side of the abdomen associated with decreased muscle tone. On retrospection he recalled that the bulging had been present on the earlier admission. The patient was advised to wear a corset. No further information is available. We interpreted these findings as indicative of abdominal muscle paresis probably due to the herpes zoster infection.

CASE 7. A 76-year-old Caucasian man noted onset of a rash and burning pain behind his right ear a week before he was admitted to the hospital February 17, 1959. On admission he had vesicular dermatitis on the right side of the neck, jaw, head and right ear. A week after admission, peripheral seventh cranial nerve paralysis appeared. This man had a three- to four-year history of arthritis of undetermined type for which he had been receiving steroids. While still in hospital for further examination and diagnosis related to arthritis, he was given electrical stimulation to the right face. About two months after admission bronchopneumonia developed and he died.

CASE 8. The patient, a 45-year-old Caucasian man, an addict to alcohol who had had left nephrectomy in 1946 for tuberculosis, was admitted to the hospital May 8, 1959 with pain and swelling in the right eye for one week followed by a vesicular eruption over the right side of the scalp, the forehead and nose. An ophthalmologist noted involvement of the right cornea. The lesions responded to local application of antibiotics, but slight ptosis of the right eyelid remained. When the patient was examined six months later the right pupil was smaller than the left and responded sluggishly to light. He also had post-herpetic neuralgia. In 1956 he had had a skull fracture for which left frontal craniotomy was done. Therefore, it is possible that the right lid ptosis and constricted pupil were due to the trauma rather than herpetic involvement of the third cranial nerve.

CASE 9. An 84-year-old Caucasian man entered the hospital September 23, 1959 with history of pain in the left side of the face followed in two weeks by blisters in the distribution of the ophthalmic division of the left fifth cranial nerve. On physical examination, ptosis of the left eyelid and a dilated left pupil which did not react to light were observed. About 10 days after admission a cere-

brovascular accident with right hemiparesis occurred. Three weeks later the patient died of myocardial infarction.

CASE 10. A 75-year-old Caucasian woman entered the hospital August 7, 1961 with severe pain of a week's duration in the right ear, neck, face, teeth, throat and shoulder. A rash over the back of the right ear had started three days before admission and had spread over the right side of the face and head. Slight weakness of the facial muscles on the right side was noted on admission examination. The rash improved on local treatment and the pain subsided. The outcome of the right facial paresis was not recorded.

CASE 11. A 46-year-old Caucasian man entered the hospital October 12, 1961 acutely ill, with pain and swelling in the left pinna, left temporal headache, nausea, dizziness, fever up to 103°F and nightly chills for six days. Three days before admission, peripheral left facial nerve palsy had begun. An audiogram showed a mild air conduction loss in the left ear. A diagnosis of Ramsay Hunt syndrome (herpes zoster oticus) was made. Antibiotics were used topically to treat a secondary infection of the external auditory canal. Within a week the acute phase subsided and partial function returned in the facial musculature. Amblyopia on the left was not attributed to the zoster. Further information was not available.

CASE 12. A 52-year-old Filipino with systemic lupus erythematosus, was admitted March 7, 1962, a week after a rash developed on the right side of his face in the distribution of the ophthalmic division of the fifth cranial nerve. The lesion was different from the butterfly rash of lupus erythematosus. The herpetic rash involved the cornea and there was also iritis of the right eye. Some drooping of the right side of the mouth, which was of unknown significance, was noted on admission examination. The hospital course was complicated by an eruption, presumably due to chloroquine which was administered for the lupus, by pulmonary disease and by bilateral parotid gland atrophy. Persistent dilatation of the right pupil, noted on this and on subsequent admissions, was attributed to the zoster.

CASE 13. A 58-year-old Caucasian woman who had diabetes was admitted to the hospital March 18, 1964 with severe pain in the left leg and a herpes zoster eruption of two weeks' duration over the first and second sacral nerve root distribution. The onset of the disease was associated with cervical and back pain, headache, chills and fever. The left knee and ankle reflexes were absent. Muscle testing showed weakness of left ankle dorsiflexion and plantarflexion and weakness of extension and flexion of the left knee. Five weeks after admission the pain was slowly resolving and the patient was able to ambulate without crutches. Unfortunately, electromyography was not done to prove the presence of lower motor neuron disease. Long-term follow-up is not available.

Comments

Various investigators have suggested that motor paralysis might be a relatively common finding in herpes zoster.[7,8,9] Grant and Rowe[8] on searching the records of the Massachusetts General Hospital uncovered five definite cases of paralysis among 101 cases of herpes zoster. However, as they recognized, this probably does not represent a true incidence of paralysis. Burgeon and coworkers[5] pointed out that herpes zoster is relatively benign and not many persons who have it enter hospitals or even seek medical care. In their report on a series of 206 cases of herpes zoster from the dermatology and ophthalmology departments of the Hospital of the University of Pennsylvania and outpatient department of Childrens' Hospital of Philadelphia, there was only a single case of extra-ocular muscle paralysis. Taterka and O'Sullivan,[19] after searching the literature in 1943, could collect only 44 cases of motor paralysis in herpes zoster, and Grant and Rowe[8] in 1961 could find reports of only 42 cases of extremity paralysis. Extra-ocular muscle involvement associated with fifth nerve gasserian ganglion zoster and facial nerve paralysis in the Ramsay Hunt syndrome seem to be more common.[5,8] More generalized, severe central nervous system involvement appears relatively rarely.[12,13,15,17]

From our own review of cases of herpes zoster from the files of the Veterans Administration Hospital, Los Angeles, it appears that true lower motor neuron lesions are uncommon, particularly in the extremities. Weakness alone, or even absence of a deep tendon reflex should not be taken as conclusive evidence of motor involvement due to the zoster. We have found that pain alone may keep a patient from responding with his full strength to manual muscle testing. Even after resolution of the pain, considerable disuse atrophy of muscle

with its associated weakness may remain for some time, giving a false impression of muscle paralysis. Absence of deep tendon reflex can be due entirely to sensory ganglion involvement. A suggestion is proposed, therefore, that electromyographic evidence of denervation be taken as the criteria of true motor paralysis. This should occur in a myotome reasonably closely associated in anatomic location with a region of typical pain and eruption of herpes zoster. Careful manual muscle testing and a neurological examination are invaluable, of course, in every case of zoster in order to disclose those in which paralysis can be suspected. Other findings of electrical testing, such as reaction of degeneration and failure to respond to faradic stimulation, are also diagnostic of true motor paralysis, but the electromyogram is more sensitive and reliable.

As in any neurological disease, the physical treatment of the patient is a most important factor. Broadbent (1866)[3] in presenting the first case of a paralytic extremity due to zoster to be recorded in modern medical literature, described the development of a "frozen shoulder" in the patient. This can be almost entirely prevented by suitable physical therapy, as was done, for example, in a more recent case reported by Parker and Ramos (1962).[14] In one of the cases herein reported (Case 1) an elderly, bedridden man was returned to ambulatory status in a walker mainly through training in the use of his remaining muscle power. The improvement in his physiological and psychological status and the improved prognosis for survival needs no elaboration. With regard to the muscle itself, galvanic electrical stimulation and massage are available to maintain the best possible condition while awaiting reinnervation. Pain may be lessened or abated by treatment with ultrasound administered over the involved dermatomes.

We have not been able to discover, either from the literature or from the cases herein reviewed, what the effect of drugs such as protamide, vitamin B_{12}, or steroids might be on the course of a motor paralysis due to herpes zoster.

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